

To Grow or Not to Grow: Specific Lipoxxygenases Control Wound-Induced Growth Restriction

As most vegetable growers know, plants have a lot of predators. Repetitive wounding events caused by herbivorous insects and necrotrophic pathogens often lead to tissue damage and growth restriction. When *Arabidopsis* (*Arabidopsis thaliana*) is subjected to recurrent wounding, leaf growth is dramatically reduced. In nature, similar stunted phenotypes can be observed in ornamental bonsai plants cultivated by skilled gardeners or when plants are attacked by herbivorous mammals or insects. In these contexts, plants overproduce jasmonates, of which the best known is jasmonic acid (JA; Browse, 2009). Inhibition of vegetative growth, as part of the wound response, occurs in wild-type plants but not in JA mutants (Zhang and Turner, 2008). Jasmonates are also required for reproductive growth, as impairment in JA synthesis or perception results in male sterility (Stintzi and Browse, 2000; Park et al., 2002).

JA synthesis is initiated in plastids where 13-lipoxygenases (LOX) add oxygen to the polyunsaturated fatty acids linolenic acid (18:3) and hexadecatrienoic acid (16:3) to form the corresponding 13-hydroperoxides, which are then cyclized to 12-oxo-phytodienoic acid, the JA precursor. In *Arabidopsis* leaves, JA biosynthesis depends on four specific 13-LOXs (LOX2, LOX3, LOX4 and LOX6). Their roles in reproductive development have been characterized; however, the function of each of these enzymes in controlling vegetative growth remains elusive.

In this issue of *Plant Physiology*, Yang et al. (2020) used a genetic approach to elucidate the contribution of each LOX to *Arabidopsis* rosette growth restriction in response to wounding. Analysis of plants with loss-of-function *lox* alleles subjected to serial wounding assays showed that LOX3 and to a lesser extent LOX4 produce the majority of JA precursors necessary for wound-induced rosette growth restriction. The authors exploited the *fatty acid oxygenation upregulated2* (*fou2*) genetic background that overaccumulates JA, which in turn triggers a transcriptomic signature similar to that produced by chewing insects (Bonaventure et al., 2007a, 2007b). The *fou2* mutant harbors a mutation in the putative voltage sensor of the *TWO PORE CHANNEL1* gene, which encodes a Ca^{2+} -permeant nonselective cation channel. Yang et al. (2020) found that the severe JA-induced growth inhibition in *fou2* is largely suppressed by mutations in *LOX3* and *LOX4*. Collectively, these data reveal the roles of LOX3 and LOX4 in rosette growth and suggest, strikingly, that the stunted

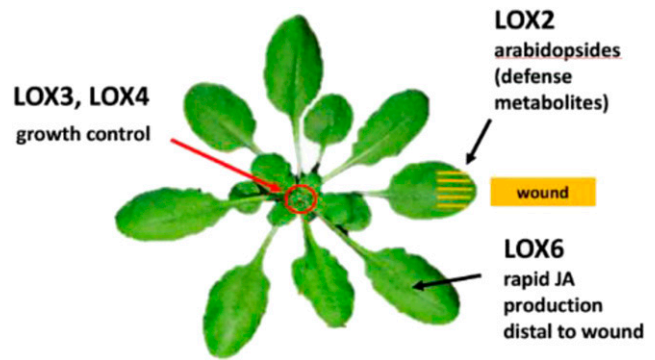


Figure 1. Schematic representation of distinct functions of 13-LOXs in *Arabidopsis*. The leaf-specific LOX2, LOX3, LOX4, and LOX6 contribute to the synthesis of jasmonate precursors and play specific roles in growth restriction in response to wounding or in defense. Overall, the specificity of LOXs seems to be dictated by their spatial and cell-specific expression. Reprinted from Yang, Lenglet-Hilfiker, et al. (2020), figure 5.

and bonsai-like wound phenotypes could be recapitulated through genetic activation of 13-LOXs in the absence of wounding (Fig. 1).

Furthermore, to test if the bonsai-like phenotype could be genetically phenocopied in unwounded tissues, Yang et al. (2020) investigated the relevance of LOX3 activation in phloem tissue, since LOX3 promoter activity spans the xylem and phloem and both these tissues are known to play major roles in leaf-to-leaf signaling (Chauvin et al., 2016; Nguyen et al., 2018). To accomplish this, Yang et al. (2020) designed a novel and elegant system where they expressed different variants of the TCP1 protein, under a phloem-specific promoter, and introduced it in the *lox* quadruple and triple mutant backgrounds with and without a functional LOX3. Phenotypic and biochemical analyses revealed that expressing a hyperactive version of TCP1 genetically activates LOX3-dependent JA production and strongly constrains rosette growth in the absence of wounding (Fig. 1).

Therefore, this new study by Yang et al. (2020) reveals how plants suffer the slings and arrows of herbivory and decide to grow or, more often, not to grow, defining specific functions for LOX3- and phloem-derived signals and providing an exciting genetic tool that will be valuable to further mechanistically elucidate the wound response and cell signaling during specific stresses.

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